(0.51 ± 0.08 vs 1.12 ± 0.31; p < 0.01) but not for D: Vlact (0.82 ± 0.32 vs 0.75 ± 0.45; p = 0.334), CSlact (0.44 ± 0.24 vs 1.12 ± 0.41; p = 0.063) and Alact (0.54 ± 0.34 vs 0.73 ± 0.41; p = 0.083). When the difference between Alact and CSlact was calculated, all pts showed lactate extraction under rest conditions and turned to lactate production during A and D infusion. Lactate production was present at least 15 min after termination of both, A and D infusion without a significant difference within the groups. The sensitivity of A and D stress echo for detecting CAD was 82% versus 84%, respectively (not significant).

Conclusion: Both drugs were highly sensitive in detecting newly developed wall motion abnormalities diagnostic for CAD. In all pts with CAD, D and A cause lactate production for at least 15 min after the end of the administration. In contrast to D, A also causes a significant elevation of peripheral venous lactate levels due to ischemia in extracardial tissue. Because of the increasing use of A in stress echocardiography the clinical importance of this new finding remains to be determined.

2:30

793-3 Assessment of Collateral Blood Flow Distribution During Myocardial Ischemia With Stress Myocardial Contrast Echocardiography in Humans

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Unlike in the animal experiments, how the collateral perfusion supply changes during myocardial stress and protects against ischemia has not been evaluated in humans. To assess the dynamic collateral function against increased myocardial oxygen demand, myocardial contrast echocardiography and rapid atrial pacing were conducted in 20 patients with angiographically significant coronary collaterals to the occluded left anterior descending coronary artery from the right coronary artery. Sonicated contrast material was injected to the right coronary artery before and during pacing to determine the peak background-subtracted contrast intensity (PCI) in the occluded bed as a parameter of collateral blood flow. Regional wall motion (RWM) was assessed by two-dimensional echocardiography, graded on a 5-point scale (0 = normal to 4 = dyskinetic). PCI before pacing ranged from 0.15 to 61.6 U, mean 14.1 U, and was significantly correlated with RWM before pacing (p < 0.05) but not with RWM during pacing. PCI was significantly decreased during pacing (p = 0.01, range 0.0 to 33.0 U, mean 7.9 U) and was significantly correlated with RWM during pacing (p < 0.01). Deterioration of RWM was not correlated with the PCI before or during pacing, but was closely related with the % decrease in PCI during pacing (p < 0.05). These findings suggested that 1) RWM was associated with collateral blood flow before or during stress, and 2) a significant decrease in collateral blood flow was associated with provocation of myocardial ischemia. In conclusion, stress MCE enabled us simultaneous assessment of dynamic changes in collateral perfusion and regional function in patients with coronary artery disease.

3:00

793-4 The Flow-Function Relationship in Patients with Chronic Coronary Artery Disease and Reduced Resting Function: a Positron Emission Tomography and 2d-Echocardiography Study with Coronary Vasodilator Stress

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In order to elucidate the flow correlates underlying the spectrum of mechanical responses of segments with resting dysfunction during vasodilator stress, 17 patients with coronary artery disease and left ventricular dysfunction underwent evaluation of regional function (2D-echocardiography) and myocardial blood flow by positron emission tomography (PET) and N13-ammonia both at rest and following dipyridamole (DIP, 0.56 mg/kg over 4'). Forty segments with resting dysfunction could be evaluated. Each segment was scored from 1 = normal/hyperkinetic to 4 = dyskinetic. Four patterns of echocardiographic response were identified: "normal" (regional wall motion score, RWMS: rest = 1, DIP = 1), n = 45; "ischemic" (RWMS: rest >1, DIP > rest), n = 9; "responders" (resting score >1, improvement ≥1 grade), n = 11; "non-responders" (resting score >1, no change), n = 20. In "normal" segments there was an increase in flow (rest = 0.8 ± 0.2 vs DIP = 1.9 ± 0.9 mL/min/g, p = 0.01) with a hyperkinetic contraction pattern. "Responders" showed an upsloping flow-function response during stress, i.e. increased function (RWMS: rest = 2.5 ± 0.5 vs DIP = 1.2 ± 0.4) and flow (rest = 0.7 ± 0.3 vs DIP = 1.9 ± 1.4 mL/min/g, p = 0.01); "non-responders" had a flat flow-function response, i.e. fixed function (RWMS: rest and DIP = 2.6 ± 0.5) and no flow increase (rest = 0.6 ± 0.2 vs DIP = 0.9 ± 0.5 mL/min/g, p = ns); "ischemic" segments exhibited a downsloping flow-function response, i.e. worsened function (RWMS: rest = 2 ± 0.5 vs DIP = 3.1 ± 0.6) and no significant flow change (rest = 0.7 ± 0.3 vs DIP = 0.8 ± 0.2 mL/min/g, p = ns). In conclusion, myocardial segments with a resting dysfunction and a contractile reserve more often exhibit a residual flow reserve, whereas segments with a fixed or worsening mechanical pattern show a flat flow response during coronary vasodilator stress.

3:15

793-5 Progression of Mitral Regurgitation and Left Ventricular Remodeling: A Quantitative Doppler Echocardiographic Study

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Mitral regurgitation (MR) due to intrinsic valvular disease causes left ventricular (LV) enlargement but the progression of the degree of regurgitation and the progression of the degree of MR and of LV remodeling occurring with time is yet unknown. In 21 patients (65 ± 7 years, 17 males) Doppler echocardiographic quantitation of MR and LV volumes and function was repeated 1.1 ± 1.3 years apart. At time of repeat evaluation no change in blood pressure (132 ± 16 vs 135 ± 11 mm/Hg, P = 0.38) and heart rate (68 ± 9 vs 72 ± 11 bpm, P = 0.19) were noted–Changes were:

Variables	First Evaluation	Second Evaluation	P
Regurgitant volume (ml)	81 ± 47	97 ± 48	0.005
Regurgitant fraction (%)	48 ± 15	55 ± 14	0.003
Effective regurgitant orifice	54 ± 34	67 ± 44	0.016
End diastolic volume (ml/m^2)	124 ± 33	134 ± 37	0.01
End systolic volume (ml/m^2)	43 ± 15	48 ± 21	0.14
Ejection fraction (%)	66 ± 7	65 ± 8	0.72
End systolic wall stress (g/cm^2)	170 ± 35	169 ± 33	0.84

The change in volume overload was highly variable (regurgitant volume changes from −13 ml to +97 ml) and showed no correlation to changes in left ventricular function (P > 0.10)

In conclusion: In chronic mitral regurgitation over a one year period: 1) marked changes in regurgitant volume but also effective regurgitant orifice are observed with 2) a mild increase in left ventricular size but no measurable change in function 3) observation of wide individual variability suggesting that regular quantitation of regurgitation is clinically relevant to assess the progression of volume overload and left ventricular remodeling in mitral regurgitation.

2:45

793-6 Near Normalization of LV Mass Following Toronto Stentless Porcine Valve Aortic Valve Replacement

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Aortic valve replacement (AVR) with stented tissue and metal prostheses results in persistingly abnormal aortic valve areas (AVA) and incomplete regression of LV hypertrophy (LVH). The Toronto Stentless Porcine valve (SPV) allows insertion of larger valve sizes and greater potential for LVH regression. The clinical and echo outcomes of 167 consecutive pts (age 62 ± 11 yrs, 115 men) undergoing SPV AVR were prospectively studied as part of an FDA protocol. Pre-operative NYHA class was 3–4 in 48%. LV mass index (LVMI, g/m2) and AVA (cm^2) were assessed prior to discharge (B), and at 3–6, 12, 24 months (m) postop. *Results:*

	B	3–6 m	12 m	24 m
Pts	167	154	137	95
LVMI	121 ± 40	111 ± 35*	104 ± 30*	98 ± 26*
AVA	2.0 ± 0.5	2.0 ± 0.5	2.2 ± 0.6*2.2|0.6*	

*p < 0.002 compared to B

Freedom from reoperation or cardiac death was 99% at 24 m; all survivors were in NYHA class 1–2. SPV design allowed insertion of larger valve sizes (27 or 29) in 65% of pts. LVMI decreased at follow-up as early as 3–6 m postoperatively. AVA increased further by 12 m and was larger than previously reported for stented or metal valves; AVA varied with valve size (23/25, 1.8 ± 0.5; 27, 2.2 ± 0.4, 29; 2.5 ± 0.7). These changes in LVMI and AVA were also evident when the study group was stratified by gender and SPV size. At 24 m near normalization of LVMI occurred for each SPV size and gender. *Conclusions:* The SPV valve permitted the use of large valve sizes, with large effective AVA, and persistent near normalization of LVMI.